

## **Appendix G: Health-Based Cost-Effectiveness of Reductions in Ambient PM<sub>2.5</sub> Associated with Illustrative PM NAAQS Attainment Strategies**

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### **G.1 Summary**

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. The Office of Management and Budget (OMB) recently issued Circular A-4 guidance on regulatory analyses, requiring federal agencies to “prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes.” Environmental quality improvements may have multiple health and ecological benefits, making application of CEA more difficult and less straightforward. For the PM NAAQS, CEA may provide a useful framework for evaluation: non-health benefits are substantial, but the majority of quantified benefits come from health effects. Therefore, EPA is including in the PM NAAQS RIA a preliminary and experimental application of one type of CEA—a modified quality-adjusted life-years (QALYs) approach.

QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA for environmental regulations. Agency concerns with the standard QALY methodology include the treatment of people with fewer years to live (the elderly); fairness to people with preexisting conditions that may lead to reduced life expectancy and reduced quality of life; and how the analysis should best account for non-health benefits, such as improved visibility.

The Institute of Medicine (a member institution of the National Academies of Science) established the Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation to assess the scientific validity, ethical implications, and practical utility of a wide range of effectiveness measures used or proposed in CEA. This committee prepared a report titled “Valuing Health for Regulatory Cost-Effectiveness Analysis” which concluded that CEA is a useful tool for assessing regulatory interventions to promote human health and safety, although not sufficient for informed regulatory decisions (Miller, Robinson, and Lawrence, 2006). They emphasized the need for additional data and methodological improvements for CEA analyses, and urged greater consistency in the reporting of assumptions, data elements, and analytic methods. They also provided a number of recommendations for the conduct of regulatory CEA analyses. EPA is evaluating these recommendations and will determine a response for upcoming analyses. For this analysis, we use the same approach that was applied in the CEA that accompanied the RIA for the Clean Air Interstate Rule.

The methodology presented in this appendix is not intended to stand as precedent either for future air pollution regulations or for other EPA regulations where it may be inappropriate. It is intended solely to demonstrate one particular approach to estimating the cost-effectiveness of reductions in ambient PM<sub>2.5</sub> in achieving improvements in public health. Reductions in ambient PM<sub>2.5</sub> likely will have other health and environmental benefits that will not be reflected in this CEA. Other EPA regulations affecting other aspects of environmental quality and public health may require additional data and models that may preclude the development of similar health-

based CEAs. A number of additional methodological issues must be considered when conducting CEAs for environmental policies, including treatment of nonhealth effects, aggregation of acute and long-term health impacts, and aggregation of life extensions and quality-of-life improvements in different populations. The appropriateness of health-based CEA should be evaluated on a case-by-case basis subject to the availability of appropriate data and models, among other factors.

Attainment of the revised PM NAAQS is expected to result in substantial reductions in potential population exposure to ambient concentrations of PM by 2020. The benefit-cost analysis presented in the RIA shows that attainment of the revised 15/35 suite of standards achieves substantial health benefits whose monetized value far exceeds costs (net benefits are over \$10 billion in 2020). Despite the risk of oversimplifying benefits, cautiously-interpreted cost-effectiveness calculations may provide further evidence of whether the costs associated with attainment strategies for the PM NAAQS are a reasonable health investment for the nation.

This analysis provides estimates of commonly used health-based effectiveness measures, including lives saved, life years saved (from reductions in mortality risk), and QALYs saved (from reductions in morbidity risk) associated with the reduction of ambient PM<sub>2.5</sub> due to illustrative attainment strategies for the revised standards and a more stringent annual standard. In addition, we use an alternative aggregate effectiveness metric, Morbidity Inclusive Life Years (MILY) to address some of the concerns about aggregation of life extension and quality-of-life impacts. It represents the sum of life years gained due to reductions in premature mortality and the QALY gained due to reductions in chronic morbidity. This measure may be preferred to existing QALY aggregation approaches because it does not devalue life extensions in individuals with preexisting illnesses that reduce quality of life. However, the MILY measure is still based on life years and thus still inherently gives more weight to interventions that reduce mortality and morbidity impacts for younger populations with higher remaining life expectancy. This analysis focuses on life extensions and improvements in quality of life through reductions in two diseases with chronic impacts: chronic bronchitis (CB) and nonfatal acute myocardial infarctions. Monte Carlo simulations are used to propagate uncertainty in several analytical parameters and characterize the distribution of estimated impacts. While the benefit-cost analysis presented in the RIA characterizes mortality impacts using a number of different sources for the PM mortality effect estimate, for this analysis, we focus on the mortality results generated using the effect estimate derived from the Pope et al. (2002) study.

Presented in three different metrics, the analysis suggests the following:

- In 2020 the illustrative attainment strategy for the revised 15/35 standards will result in:
  - 2,500 (95% CI: 1,000 – 4,100) premature deaths avoided, or
  - 26,000 (95% CI: 18,000 – 34,000) life years gained (discounted at 3 percent), or
  - 43,000 (95% CI: 28,000 – 62,000) MILYs gained (discounted at 3 percent).
- In 2020, the illustrative attainment strategy for the more stringent 14/35 standards will result in:
  - 4,400 (95% CI: 1,700 – 7,100) premature deaths avoided, or

- 45,000 (95% CI: 32,000 – 59,000) life years gained (discounted at 3 percent), or
- 75,000 (95% CI: 48,000 – 107,000) MILYs gained (discounted at 3 percent).
- Using a 7 percent discount rate, mean discounted life years gained are 16,000 for the revised 15/35 standards and 29,000 for the alternative 14/35 standards; mean MILYs gained are 28,000 for the 15/35 standards and 49,000 for the alternative 14/35 standards. (The estimates of premature deaths avoided are not affected by the discount rate.)
- The associated reductions in CB and nonfatal acute myocardial infarctions will reduce medical costs by approximately \$680 million for the 15/35 scenario and \$1,200 million for the 14/35 scenario based on a 3 percent discount rate, or \$520 million for the 15/35 scenario and \$940 million for the 14/35 scenario based on a 7 percent discount rate.
- Other health and visibility benefits are valued at \$530 million for the 15/35 scenario and \$1,100 million for the 14/35 scenario.

Direct private compliance costs for the 15/35 attainment strategy, including the extrapolated costs of full attainment in California and Salt Lake City are \$5.4 billion, incremental to attainment of the current 15/65 standards in 2020. Full attainment costs for the 14/35 attainment strategy are \$7.0 billion incremental to attainment of the current 15/65 standards. Based on these costs, the incremental cost effectiveness (net of cost of illness and other health and visibility benefits) of the 15/35 attainment strategy relative to attainment of the current standards is \$98,000/MILY using a 3 percent discount rate and \$160,000/MILY using a 7 percent discount rate. Incremental cost effectiveness of the 14/35 attainment strategy relative to attainment of the current standards is \$60,000/MILY using a 3 percent discount rate and \$100,000/MILY using a 7 percent discount rate. The incremental cost effectiveness of the attainment strategy for the alternative 14/35 standards relative to the attainment strategy for the revised 15/35 standards is \$17,000/MILY using a 3 percent discount rate and \$29,000 using a 7 percent discount rate. The relatively smaller incremental cost per MILY associated with the attainment strategy for the alternative 14/35 standards is primarily due to the regional control strategies implemented in the Eastern U.S. (which affect a much larger population), and the fact that much of the cost of both the 15/35 and 14/35 attainment strategies is due to the high estimates of costs of attaining the daily standard of 35  $\mu\text{g}/\text{m}^3$  in California. See Chapters 4 and 5 of this RIA for more discussion of the control strategies and cost estimates.

## **G.2 Introduction**

Analyses of environmental regulations have typically used benefit-cost analysis to characterize impacts on social welfare. Benefit-cost analyses allow for aggregation of the benefits of reducing mortality risks with other monetized benefits of reducing air pollution, including acute and chronic morbidity, and nonhealth benefits such as improved visibility. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. However, alternative paradigms such as CEA and CUA analyses may also provide useful insights. CEA involves estimation of the costs per unit of benefit (e.g., lives or life years saved). CUA is a special type of CEA using preference-based measures of effectiveness, such as QALYs.

CEA and CUA are most useful for comparing programs that have similar goals, for example, alternative medical interventions or treatments that can save a life or cure a disease. They are less readily applicable to programs with multiple categories of benefits, such as those reducing ambient air pollution, because the cost-effectiveness calculation is based on the quantity of a single benefit category. In other words, we cannot readily convert improvements in nonhealth benefits such as visibility to a health metric such as life years saved. For these reasons, environmental economists prefer to present results in terms of monetary benefits and net benefits.

However, QALY-based CUA has been widely adopted within the health economics literature (Neumann, 2003; Gold et al., 1996) and in the analysis of public health interventions (US FDA, 2004). QALY-based analyses have not been as accepted in the environmental economics literature because of concerns about the theoretical consistency of QALYs with individual preferences (Hammitt, 2002), treatment of nonhuman health benefits, and a number of other factors (Freeman, Hammitt, and De Civita, 2002). For environmental regulations, benefit-cost analysis has been the preferred method of choosing among regulatory alternatives in terms of economic efficiency. Recently several academic analyses have proposed the use of life years-based benefit-cost or CEAs of air pollution regulations (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003; Rabl, 2003; Carrothers, Evans, and Graham, 2002). In addition, the World Health Organization has adopted the use of disability-adjusted life years, a variant on QALYs, to assess the global burden of disease due to different causes, including environmental pollution (Murray et al., 2002; de Hollander et al., 1999).

Recently, the U.S. OMB (Circular A-4, 2003) issued new guidance requiring federal agencies to provide both CEA and benefit-cost analyses for major regulations. The OMB Circular A-4 directs agencies to “prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes.” We are including a CEA for the illustrative PM NAAQS attainment strategies to illustrate one potential approach for conducting a CEA. EPA is still evaluating the appropriate methods for CEA for environmental regulations with multiple outcomes.

The methodology presented in this appendix is not intended to stand as precedent either for future air pollution regulations or for other EPA regulations governing water, solid waste, or other regulatory objectives. It is intended solely to demonstrate one particular approach to estimating the effectiveness of reductions in ambient PM<sub>2.5</sub> in achieving improvements in public health. This analysis focuses on effectiveness measured by improvements in life expectancy and reductions in the incidence of two diseases with chronic impacts on quality of life: CB and nonfatal acute myocardial infarctions. Other EPA regulations affecting other aspects of environmental quality and public health may require additional data and models that may preclude the development of similar QALY-based analyses. The appropriateness of QALY-based CEA should be evaluated on a case-by-case basis subject to the availability of appropriate data and models.

Preparation of a CEA requires identification of an appropriate measure of rule effectiveness. Given the significant impact of reductions in ambient PM<sub>2.5</sub> on reductions in the risk of mortality, lives saved is an important measure of effectiveness. However, one of the ongoing

controversies in health impact assessment regards whether reductions in mortality risk should be reported and valued in terms of statistical lives saved or in terms of statistical life years saved. Life years saved measures differentiate among premature mortalities based on the remaining life expectancy of affected individuals. In general, under the life years approach, older individuals will gain fewer life years than younger individuals for the same reduction in mortality risk during a given time period, making interventions that benefit older individuals seem less beneficial relative to similar interventions benefiting younger individuals. A further complication in the debate is whether to apply quality adjustments to life years lost. Under this approach, individuals with preexisting health conditions would have fewer QALYs lost relative to healthy individuals for the same loss in life expectancy, making interventions that primarily benefit individuals with poor health seem less beneficial to similar interventions affecting primarily healthy individuals.

In addition to substantial mortality risk reduction benefits, strategies for attaining the revised PM NAAQS will also result in significant reductions in chronic and acute morbidity. Several approaches have been developed to incorporate both morbidity and mortality into a single effectiveness metric. The most common of these is the QALY approach, which expresses all morbidity and mortality impacts in terms of quality of life multiplied by the duration of time with that quality of life. The QALY approach has some appealing characteristics. For example, it can account for morbidity effects as well as losses in life expectancy without requiring the assignment of dollar values to calculate total benefits. By doing so it provides an alternative framework to benefit-cost analysis for aggregating quantitative measures of health impacts.

While used extensively in the economic evaluation of medical interventions (Gold et al., 1996), QALYs have not been widely used in evaluating environmental health regulations. A number of specific issues arise with the use of QALYs in evaluating environmental programs that affect a broad and heterogeneous population and that provide both health and nonhealth benefits. The U.S. Public Health Service report on cost-effectiveness in health and medicine notes the following:

For decisions that involve greater diversity in interventions and the people to whom they apply, cost-effectiveness ratios continue to provide essential information, but that information must, to a greater degree, be evaluated in light of circumstances and values that cannot be included in the analysis. Individuals in the population will differ widely in their health and disability before the intervention, or in age, wealth, or other characteristics, raising questions about how society values gains for the more and less health, for young and old, for rich and poor, and so on. The assumption that all QALYs are of equal value is less likely to be reasonable in this context. (Gold et al., 1996, p. 11)

Use of QALYs as a measure of effectiveness for environmental regulations is still developing, and while this analysis provides one framework for using QALYs to evaluate environmental regulations, there are clearly many issues, both scientific and ethical, that need to be addressed with additional research. The Institute of Medicine panel evaluating QALYs and other effectiveness measures prepared a report titled “Valuing Health for Regulatory Cost-Effectiveness Analysis” which concluded that “the QALY is the best measure at present on which to standardize Health Adjusted Life Year estimation because of its widespread use,

flexibility, and relative simplicity” (Miller, Robinson, and Lawrence, 2006). EPA is evaluating this recommendation and will determine a response for upcoming analyses. For this analysis, for reasons discussed in the text, we use the same MILY approach that was applied in the CEA that accompanied the RIA for the Clean Air Interstate Rule.

This appendix presents cost-effectiveness methodologies for evaluating programs such as attainment strategies for the revised PM NAAQS that are intended to reduce ambient PM<sub>2.5</sub> starting from the standard QALY literature and seeking a parallel structure to benefit-cost analysis in the use of air quality and health inputs (see Hubbell [2004a] for a discussion of some of the issues that arise in comparing QALY and benefit-cost frameworks in analyzing air pollution impacts). For the purposes of this analysis, we calculate effectiveness using several different metrics, including lives prolonged, life years gained, and modified QALYs. For the life years and QALY-type approaches, we use life table methods to calculate the change in life expectancy expected to result from changes in mortality risk from PM. We use existing estimates of preferences for different health states to obtain QALY weights for morbidity endpoints associated with air pollution. In general, consistent with the Gold et al. (1996) recommendations, we use weights obtained from a societal perspective when available. We explore several different sources for these weights to characterize some of the potential uncertainty in the QALY estimates. We follow many of the principles of the reference case analysis as defined in Gold et al. (1996), although in some cases we depart from the reference case approach when data limitations require us to do so (primarily in the selection of quality-of-life weights for morbidity endpoints). We also depart from the reference case (and the recommendations of the IOM report) in the method of combining life expectancy and quality-of-life gains.

Results in most tables are presented only at a discount rate of 3 percent, rather than at both 3 percent and 7 percent as recommended in EPA and OMB guidance. This is strictly for ease of presentation. Aggregate results at 7 percent are presented in the summary, and the impact of using a 7 percent discount rate instead of 3 percent rate is summarized in a sensitivity analysis.

Monte Carlo simulation methods are used to propagate uncertainty in several of the model parameters throughout the analysis. We characterize overall uncertainty in the results with 95 percent confidence intervals based on the Monte Carlo simulations. In addition, we examine the impacts of changing key parameters, such as the discount rate, on the effectiveness measures and the cost-effectiveness metrics.

The remainder of this appendix provides an overview of the key issues involved in life year- and QALY-based approaches for evaluating the health impacts of air pollution regulations, provides detailed discussions of the steps required for each type of effectiveness calculation, and presents the CEA for the PM NAAQS illustrative attainment strategies. Section G.3 introduces the various effectiveness measures and discusses some of the assumptions required for each. Section G.4 details the methodology used to calculate changes in life years and quality adjustments for mortality and morbidity endpoints. Section G.5 provides the results for the illustrative attainment strategies for the revised and more stringent alternative PM NAAQS and discusses their implications for cost-effectiveness of these attainment strategies.

### G.3 Effectiveness Measures

Three major classes of benefits are associated with reductions in air pollution: mortality, morbidity, and nonhealth (welfare). For the purposes of benefit-cost analysis, EPA has presented mortality-related benefits using estimates of avoided premature mortalities, representing the cumulative result of reducing the risk of premature mortality from long-term exposure to PM<sub>2.5</sub> for a large portion of the U.S. population. Morbidity benefits have been characterized by numbers of new incidences avoided for chronic diseases such as CB, avoided admissions for hospitalizations associated with acute and chronic conditions, and avoided days with symptoms for minor illnesses. Nonhealth benefits are characterized by the monetary value of reducing the impact (e.g., the dollar value of improvements in visibility at national parks).

For the purposes of CEA, we focus the effectiveness measure on the quantifiable health impacts of the reduction in PM<sub>2.5</sub>. Treatment of nonhealth benefits is important and is discussed in some detail later in this section. If the main impact of interest is reductions in mortality risk from air pollution, the effectiveness measures are relatively straightforward to develop. Mortality impacts can be characterized similar to the benefits analysis, by counting the number of premature mortalities avoided, or can be characterized in terms of increases in life expectancy or life years.<sup>1</sup> Estimates of premature mortality have the benefit of being relatively simple to calculate, are consistent with the benefit-cost analysis, and do not impose additional assumptions on the degree of life shortening. However, some have argued that counts of premature mortalities avoided are problematic because a gain in life of only a few months would be considered equivalent to a gain of a many life years, and the true effectiveness of an intervention is the gain in life expectancy or life years (Rabl, 2003; Miller and Hurley, 2003).

Calculations of changes in life years and life expectancy can be accomplished using standard life table methods (Miller and Hurley, 2003). However, the calculations require assumptions about the baseline mortality risks for each age cohort affected by air pollution. A general assumption may be that air pollution mortality risks affect the general mortality risk of the population in a proportional manner. However, some concerns have been raised that air pollution affects mainly those individuals with preexisting cardiovascular and respiratory disease, who may have reduced life expectancy relative to the general population. This issue is explored in more detail below.

Air pollution is also associated with a number of significant chronic and acute morbidity endpoints. Failure to consider these morbidity effects may understate the cost-effectiveness of air pollution regulations or give too little weight to reductions in particular pollutants that have large morbidity impacts but no effect on life expectancy. The QALY approach explicitly incorporates morbidity impacts into measures of life years gained and is often used in health economics to assess the cost-effectiveness of medical spending programs (Gold et al., 1996).

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<sup>1</sup> Life expectancy is an *ex ante* concept, indicating the impact on an entire population's expectation of the number of life years they have remaining, before knowing which individuals will be affected. Life expectancy thus incorporates both the probability of an effect and the impact of the effect if realized. Life years is an *ex post* concept, indicating the impact on individuals who actually die from exposure to air pollution. Changes in population life expectancy will always be substantially smaller than changes in life years per premature mortality avoided, although the total life years gained in the population will be the same. This is because life expectancy gains average expected life years gained over the entire population, while life years gained measures life years gained only for those experiencing the life extension.

Using a QALY rating system, health quality ranges from 0 to 1, where 1 may represent full health, 0 death, and some number in between (e.g., 0.8) an impaired condition. QALYs thus measure morbidity as a reduction in quality of life over a period of life. QALYs assume that duration and quality of life are equivalent, so that 1 year spent in perfect health is equivalent to 2 years spent with quality of life half that of perfect health. QALYs can be used to evaluate environmental rules under certain circumstances, although some very strong assumptions (detailed below) are associated with QALYs. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommended using QALYs when evaluating medical and public health programs that primarily reduce both mortality and morbidity (Gold et al., 1996). Although there are significant nonhealth benefits associated with air pollution regulations, over 90 percent of quantifiable monetized benefits are health-related, as is the case with the attainment strategies for the PM NAAQS. Thus, it can be argued that QALYs are more applicable for these types of regulations than for other environmental policies. However, the value of nonhealth benefits should not be ignored. As discussed below, we have chosen to subtract the value of nonhealth benefits from the costs in the numerator of the cost-effectiveness ratio.

In the following sections, we lay out a phased approach to describing effectiveness. We begin by discussing how the life-extending benefits of air pollution reductions are calculated, and then we incorporate morbidity effects using the QALY approach. We also introduce an alternative aggregated health metric, Morbidity Inclusive Life Years (MILY) to address some of the ethical concerns about aggregating life extension impacts in populations with preexisting disabling conditions.

The use of QALYs is predicated on the assumptions embedded in the QALY analytical framework. As noted in the QALY literature, QALYs are consistent with the utility theory that underlies most of economics only if one imposes several restrictive assumptions, including independence between longevity and quality of life in the utility function, risk neutrality with respect to years of life (which implies that the utility function is linear), and constant proportionality in trade-offs between quality and quantity of life (Pliskin, Shepard, and Weinstein, 1980; Bleichrodt, Wakker, and Johannesson, 1996). To the extent that these assumptions do not represent actual preferences, the QALY approach will not provide results that are consistent with a benefit-cost analysis based on the Kaldor-Hicks criterion.<sup>2</sup> Even if the assumptions are reasonably consistent with reality, because QALYs represent an average valuation of health states rather than the sum of societal WTP, there are no guarantees that the option with the highest QALY per dollar of cost will satisfy the Kaldor-Hicks criterion (i.e., generate a potential Pareto improvement [Garber and Phelps, 1997]).

Benefit-cost analysis based on WTP is not without potentially troubling underlying structures as well, incorporating ability to pay (and thus the potential for equity concerns) and the notion of consumer sovereignty (which emphasizes wealth effects). Table G-1 compares the two approaches across a number of parameters. For the most part, WTP allows parameters to be determined empirically, while the QALY approach imposes some conditions *a priori*.

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<sup>2</sup> The Kaldor-Hicks efficiency criterion requires that the “winners” in a particular case be potentially able to compensate the “losers” such that total societal welfare improves. In this case, it is sufficient that total benefits exceed total costs of the regulation. This is also known as a potential Pareto improvement, because gains could be allocated such that at least one person in society would be better off while no one would be worse off.



**Table G-1: Comparison of QALY and WTP Approaches**

<i>Parameter</i>	<i>QALY</i>	<i>WTP</i>
Risk aversion	Risk neutral	Empirically determined
Relation of duration and quality	Independent	Empirically determined
Proportionality of duration/ quality trade-off	Constant	Variable
Treatment of time/age in utility function	Utility linear in time	Empirically determined
Preferences	Community/Individual	Individual
Source of preference data	Stated	Revealed and stated
Treatment of income and prices	Not explicitly considered	Constrains choices

#### **G.4 Changes in Premature Death, Life Years, and Quality of Life**

To generate health outcomes, we used the same framework as for the benefit-cost analysis described in Chapter 5. For convenience, we summarize the basic methodologies here. For more details, see Chapter 5 and the BenMAP user's manual (<http://www.epa.gov/ttn/ecas/benmodels.html>).

BenMAP uses health impact functions to generate changes in the incidence of health effects. Health impact functions are derived from the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics like CDC), the affected population, and the estimated change in the relevant PM summary measure.

A typical health impact function might look like this:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1),$$

where  $y_0$  is the baseline incidence, equal to the baseline incidence rate times the potentially affected population;  $\beta$  is the effect estimate; and  $\Delta x$  is the estimated change in  $PM_{2.5}$ . There are other functional forms, but the basic elements remain the same.

##### *G.4.1 Calculating Reductions in Premature Deaths*

As in several recent air pollution health impact assessments (e.g., Kunzli et al., 2000; EPA, 2004), we focus on the prospective cohort long-term exposure studies in deriving the health impact function for the estimate of premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli et al., 2001; NRC, 2002). We selected an effect estimate from the extended analysis of the ACS cohort (Pope et al., 2002). This latest re-analysis of the ACS cohort data provides additional refinements to the analysis of PM-related mortality by (a) extending the follow-up period for the ACS study

subjects to 16 years, which triples the size of the mortality data set; (b) substantially increasing exposure data, including consideration for cohort exposure to PM<sub>2.5</sub> following implementation of PM<sub>2.5</sub> standard in 1999; (c) controlling for a variety of personal risk factors including occupational exposure and diet; and (d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates, including the possibility of spatial autocorrelation of survival times in communities located near each other. The effect estimate from Pope et al. (2002) quantifies the relationship between annual mean PM<sub>2.5</sub> levels and all-cause mortality in adults 30 and older. We selected the effect estimate estimated using the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979–1984 and 1999–2000 PM<sub>2.5</sub> levels. The effect estimate from this study is 0.0058, which is equivalent to a relative risk of 1.06 for a 10 µg change in PM<sub>2.5</sub>. Although there are other cohort-based studies of the relationship between PM<sub>2.5</sub> and mortality, none provide the same level of population and geographic coverage as the ACS study.

Age, cause, and county-specific mortality rates were obtained from CDC for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at <http://wonder.cdc.gov/>. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups.

The reductions in incidence of premature mortality within each age group associated with the illustrative attainment strategies for the revised and more stringent alternative PM NAAQS in 2020 are summarized in Table G-2.

#### *G.4.2 Calculating Changes in Life Years from Direct Reductions in PM<sub>2.5</sub>-Related Mortality Risk*

To calculate changes in life years associated with a given change in air pollution, we used a life table approach coupled with age-specific estimates of reductions in premature mortality. We began with the complete unabridged life table for the United States in 2000, obtained from CDC (CDC, 2002). For each 1-year age interval (e.g., zero to one, one to two) the life table provides estimates of the baseline probability of dying during the interval, person years lived in the interval, and remaining life expectancy. From this unabridged life table, we constructed an abridged life table to match the age intervals for which we have predictions of changes in incidence of premature mortality. We used the abridgement method described in CDC (2002). Table G-3 presents the abridged life table for 10-year age intervals for adults over 30 (to match the Pope et al. [2002] study population). Note that the abridgement actually includes one 5-year interval, covering adults 30 to 34, with the remaining age intervals covering 10 years each. This is to provide conformity with the age intervals available for mortality rates.

**Table G-2: Estimated Reduction in Incidence of All-cause Premature Mortality Associated with Illustrative Attainment Strategies for the Revised and More Stringent Alternative PM NAAQS in 2020**

<i>Reduction in All-Cause Premature Mortality (95% CI)</i>		
<i>Age Interval</i>	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>
30 – 34	25 (8 – 41)	40 (13 – 68)
35 – 44	76 (25 – 130)	120 (39 – 210)
45 – 54	150 (48 – 250)	250 (80 – 420)
55 – 64	350 (110 – 590)	610 (200 – 1,000)
65 – 74	530 (170 – 890)	970 (310 – 1,600)
75 – 84	610 (200 – 1,000)	1,100 (350 – 1,800)
85+	810 (260 – 1,400)	1,300 (430 – 2,300)
Total	2,500 (820 – 4,300)	4,400 (1,400 – 7,400)

From the abridged life table (Table G-3), we obtained the remaining life expectancy for each age cohort, conditional on surviving to that age. This is then the number of life years lost for an individual in the general population dying during that age interval. This information can then be combined with the estimated number of premature deaths in each age interval calculated with BenMAP (see previous subsection). Total life years gained will then be the sum of life years gained in each age interval:

$$TotalLife\ Years = \sum_{i=1}^N LE_i \times M_i ,$$

where  $LE_i$  is the remaining life expectancy for age interval  $i$ ,  $M_i$  is the change in incidence of mortality in age interval  $i$ , and  $N$  is the number of age intervals.

For the purposes of determining cost-effectiveness, it is also necessary to consider the time-dependent nature of the gains in life years. Standard economic theory suggests that benefits occurring in future years should be discounted relative to benefits occurring in the present. OMB and EPA guidance suggest discount rates of three and seven percent. As noted earlier, we present gains in future life years discounted at 3 percent. Results based on 7 percent are included in the summary and the overall impact of a 7 percent rate is summarized in Table G-16. Selection of a 3 percent discount rate is also consistent with recommendations from the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine (Gold et al., 1996).

**Table G-3: Abridged Life Table for the Total Population, United States, 2000**

Age Interval		Probability of Dying Between Ages x to x+1	Number Surviving to Age x	Number Dying Between Ages x to x+1	Person Years Lived Between Ages x to x+1	Total Number of Person Years Lived Above Age x	Expectation of Life at Age x
Start Age	End Age	$q_x$	$l_x$	$d_x$	$L_x$	$T_x$	$e_x$
30	35	0.00577	97,696	564	487,130	4,723,539	48.3
35	45	0.01979	97,132	1,922	962,882	4,236,409	43.6
45	55	0.04303	95,210	4,097	934,026	3,273,527	34.4
55	65	0.09858	91,113	8,982	872,003	2,339,501	25.7
65	75	0.21779	82,131	17,887	740,927	1,467,498	17.9
75	85	0.45584	64,244	29,285	505,278	726,571	11.3
85	95	0.79256	34,959	27,707	196,269	221,293	6.3
95	100	0.75441	7,252	5,471	20,388	25,024	3.5
100+		1.00000	1,781	1,781	4,636	4,636	2.6

Discounted total life years gained is calculated as follows:

$$\text{Discounted LY} = \int_0^{LE} e^{-rt} dt$$

where  $r$  is the discount rate, equal to 0.03 in this case,  $t$  indicates time, and  $LE$  is the life expectancy at the time when the premature death would have occurred. Life years are further discounted to account for the lag between the reduction in ambient  $PM_{2.5}$  and the reduction in mortality risk. We use the same 20-year segmented lag structure that is used in the benefit-cost analysis (see Chapter 5).

The most complete estimate of the impacts of  $PM_{2.5}$  on life years is calculated using the Pope et al. (2002) C-R function relating all-cause mortality in adults 30 and over with ambient  $PM_{2.5}$  concentrations averaged over the periods 1979–1983 and 1999–2000. Use of all-cause mortality is appropriate if there are no differences in the life expectancy of individuals dying from air pollution-related causes and those dying from other causes. The argument that long-term exposure to  $PM_{2.5}$  may affect mainly individuals with serious preexisting illnesses is not supported by current empirical studies. For example, the Krewski et al. (2000) ACS reanalysis suggests that the mortality risk is no greater for those with preexisting illness at time of enrollment in the study. Life expectancy for the general population in fact includes individuals with serious chronic illness. Mortality rates for the general population then reflect prevalence of chronic disease, and as populations age the prevalence of chronic disease increases.

The only reason one might use a lower life expectancy is if the population at risk from air pollution was limited solely to those with preexisting disease. Also, note that the OMB Circular A-4 notes that “if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimate number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts.” As such, use of a general population life expectancy is preferred over disability-specific life expectancies. Our primary life years calculations are thus consistent with the concept of not penalizing individuals with disabling chronic health conditions by assessing them reduced benefits of mortality risk reductions.

For this analysis, direct impacts on life expectancy are measured only through the estimated change in mortality risk based on the Pope et al. (2002) C-R function. The SAB-HES has advised against including additional gains in life expectancy due to reductions in incidence of chronic disease or nonfatal heart attacks (EPA-SAB-COUNCIL-ADV-04-002). Although reductions in these endpoints are likely to result in increased life expectancy, the HES has suggested that the cohort design and relatively long follow-up period in the Pope et al. study should capture any life-prolonging impacts associated with those endpoints. Impacts of CB and nonfatal heart attacks on quality of life will be captured separately in the QALY calculation as years lived with improved quality of life. The methods for calculating this benefit are discussed below.

#### G.4.2.1 Should Life Years Gained Be Adjusted for Initial Health Status?

The methods outlined above provide estimates of the total number of life years gained in a population, regardless of the quality of those life years, or equivalently, assuming that all life years gained are in perfect health. In some CEAs (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003), analysts have adjusted the number of life years gained to reflect the fact that 1) the general public is not in perfect health and thus “healthy” life years are less than total life years gained and 2) those affected by air pollution may be in a worse health state than the general population and therefore will not gain as many “healthy” life years adjusted for quality, from an air pollution reduction. This adjustment, which converts life years gained into QALYs, raises a number of serious ethical issues. Proponents of QALYs have promoted the nondiscriminatory nature of QALYs in evaluating improvements in quality of life (e.g., an improvement from a score of 0.2 to 0.4 is equivalent to an improvement from 0.8 to 1.0), so the starting health status does not affect the evaluation of interventions that improve quality of life. However, for life-extending interventions, the gains in QALY will be directly proportional to the baseline health state (e.g., an individual with a 30-year life expectancy and a starting health status of 0.5 will gain exactly half the QALYs of an individual with the same life expectancy and a starting health status of 1.0 for a similar life-extending intervention). This is troubling because it imposes an additional penalty for those already suffering from disabling conditions. Brock (2002) notes that “the problem of disability discrimination represents a deep and unresolved problem for resource prioritization.”

OMB (2003) has recognized this issue in their Circular A-4 guidance, which includes the following statement:

When CEA is performed in specific rulemaking contexts, you should be prepared to make appropriate adjustments to ensure fair treatment of all segments of the population. Fairness is important in the choice and execution of effectiveness measures. For example, if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves the lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimated number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts. More generally, when numeric adjustments are made for life expectancy or quality of life, analysts should prefer use of population averages rather than information derived from subgroups dominated by a particular demographic or income group. (p. 13)

This suggests two adjustments to the standard QALY methodology: one adjusting the relevant life expectancy of the affected population, and the other affecting the baseline quality of life for the affected population.

In addition to the issue of fairness, potential measurement issues are specific to the air pollution context that might argue for caution in applying quality-of-life adjustments to life years gained due to air pollution reductions. A number of epidemiological and toxicological studies link exposure to air pollution with chronic diseases, such as CB and atherosclerosis (Abbey et al., 1995; Schwartz, 1993; Suwa et al., 2002). If these same individuals with chronic disease caused by exposure to air pollution are then at increased risk of premature death from air pollution, there is an important dimension of “double jeopardy” involved in determining the correct baseline for assessing QALYs lost to air pollution (see Singer et al. [1995] for a broader discussion of the double-jeopardy argument).

Analyses estimating mortality from acute exposures that ignore the effects of long-term exposure on morbidity may understate the health impacts of reducing air pollution. Individuals exposed to chronically elevated levels of air pollution may realize an increased risk of death and chronic disease throughout life. If at some age they contract heart (or some other chronic) disease as a result of the exposure to air pollution, they will from that point forward have both reduced life expectancy and reduced quality of life. The benefit to that individual from reducing lifetime exposure to air pollution would be the increase in life expectancy plus the increase in quality of life over the full period of increased life expectancy. If the QALY loss is determined based on the underlying chronic condition and life expectancy without regard to the fact that the person would never have been in that state without long-term exposure to elevated air pollution, then the person is placed in double jeopardy. In other words, air pollution has placed more people in the susceptible pool, but then we penalize those people in evaluating policies by treating their subsequent deaths as less valuable, adding insult to injury, and potentially downplaying the importance of life expectancy losses due to air pollution. If the risk of chronic disease and risk of death are considered together, then there is no conceptual problem with measuring QALYs, but this has not been the case in recent applications of QALYs to air pollution (Carrothers,

Evans, and Graham, 2002; Coyle et al., 2003). The use of QALYs thus highlights the need for a better understanding of the relationship between chronic disease and long-term exposure and suggests that analyses need to consider morbidity and mortality jointly, rather than treating each as a separate endpoint (this is an issue for current benefit-cost approaches as well).

Because of the fairness and measurement concerns discussed above, for the purposes of this analysis, we do not reduce the number of life years gained to reflect any differences in underlying health status that might reduce quality of life in remaining years. Thus, we maintain the assumption that all direct gains in life years resulting from mortality risk reductions will be assigned a weight of 1.0. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that “since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY” (Gold et al., 1996). However, for the purposes of this analysis, we propose an alternative to the traditional aggregate QALY metric that keeps separate quality adjustments to life expectancy and gains in life expectancy. As such, we do not make any adjustments to life years gained to reflect the less than perfect health of the general population. Gains in quality of life will be addressed as they accrue because of reductions in the incidence of chronic diseases. This is an explicit equity choice in the treatment of issues associated with quality-of-life adjustments for increases in life expectancy that still capitalizes on the ability of QALYs to capture both morbidity and mortality impacts in a single effectiveness measure.

## **G.5 Calculating Changes in the Quality of Life Years (Morbidity)**

In addition to directly measuring the quantity of life gained, measured by life years, it may also be informative to measure gains in the quality of life. Reducing air pollution also leads to reductions in serious illnesses that affect quality of life. These include CB and cardiovascular disease, for which we are able to quantify changes in the incidence of nonfatal heart attacks. To capture these important benefits in the measure of effectiveness, they must first be converted into a life-year equivalent so that they can be combined with the direct gains in life expectancy.

For this analysis, we developed estimates of the QALYs gained from reductions in the incidence of CB and nonfatal heart attacks associated with reductions in ambient PM<sub>2.5</sub>. In general, QALY calculations require four elements:

1. the estimated change in incidence of the health condition,
2. the duration of the health condition,
3. the quality-of-life weight with the health condition, and
4. the quality-of-life weight without the health condition (i.e., the baseline health state).

The first element is derived using the health impact function approach. The second element is based on the medical literature for each health condition. The third and fourth elements are derived from the medical cost-effectiveness and cost-utility literature. In the following two subsections, we discuss the choices of elements for CB and nonfatal heart attacks.

The preferred source of quality-of-life weights are those based on community preferences, rather than patient or clinician ratings (Gold et al., 1996). Several methods are used to estimate quality-

of-life weights. These include rating scale, standard gamble, time trade-off, and person trade-off approaches (Gold, Stevenson, and Fryback, 2002). Only the standard gamble approach is completely consistent with utility theory. However, the time trade-off method has also been widely applied in eliciting community preferences (Gold, Stevenson, and Fryback, 2002).

Quality-of-life weights can be directly elicited for individual specific health states or for a more general set of activity restrictions and health states that can then be used to construct QALY weights for specific conditions (Horsman et al., 2003; Kind, 1996). For this analysis, we used weights based on community-based preferences, using time trade-off or standard gamble when available. In some cases, we used patient or clinician ratings when no community preference-based weights were available. Sources for weights are discussed in more detail below. Table G-4 summarizes the key inputs for calculating QALYs associated with chronic health endpoints.

#### *G.5.1 Calculating QALYs Associated with Reductions in the Incidence of Chronic Bronchitis*

CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). For gains in quality of life resulting from reduced incidences of PM-induced CB, discounted QALYs are calculated as

$$DISCOUNTED\ QALYGAINED = \sum_i \Delta CB_i \times D_i^* \times (w_i - w_i^{CB})$$

where  $\Delta CB_i$  is the number of incidences of CB avoided in age interval  $i$ ,  $w_i$  is the average QALY weight for age interval  $i$ ,  $w_i^{CB}$  is the QALY weight associated with CB,  $D_i^*$  is the discounted duration of life with CB for individuals with onset of disease in age interval  $i$ , equal to  $\int_{t=1}^{D_i} e^{-rt} dt$ , where  $D_i$  is the duration of life with CB for individuals with onset of disease in age interval  $i$ .

A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because this analysis focuses on the impacts of reducing ambient PM<sub>2.5</sub>, only the Abbey et al. (1995) study is used, because it is the only study focusing on the relationship between PM<sub>2.5</sub> and new incidences of CB. The number of cases of CB in each age interval is derived from applying the impact function from Abbey et al. (1995), to the population in each age interval with the appropriate baseline incidence rate.<sup>3</sup> The effect estimate from the Abbey et al. (1995) study is 0.0137, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.15 for a 10 µg change in PM<sub>2.5</sub>. Table G-5 presents the estimated reduction in new incidences of CB associated with the illustrative PM NAAQS attainment strategies.

<sup>3</sup> Prevalence rates for CB were obtained from the 1999 National Health Interview Survey (American Lung Association, 2002). Prevalence rates were available for three age groups: 18–44, 45–64, and 65 and older. Prevalence rates per person for these groups were 0.0367 for 18–44, 0.0505 for 45–64, and 0.0587 for 65 and older. The incidence rate for new cases of CB (0.00378 per person) was taken directly from Abbey et al. (1995).



**Table G-4: Summary of Key Parameters Used in QALY Calculations for Chronic Disease Endpoints**

<i>Parameter</i>	<i>Value(s)</i>	<i>Source(s)</i>
Discount rate	0.03 (0.07 sensitivity analysis)	Gold et al. (1996), U.S. EPA (2000), U.S. OMB (2003)
Quality of life preference score for chronic bronchitis	0.5 – 0.7	Triangular distribution centered at 0.7 with upper bound at 0.9 (Vos, 1999a) (slightly better than a mild/moderate case) and a lower bound at 0.5 (average weight for a severe case based on Vos [1999a] and Smith and Peske [1994])
Duration of acute phase of acute myocardial infarction (AMI)	5.5 days – 22 days	Uniform distribution with lower bound based on average length of stay for an AMI (AHRQ, 2000) and upper bound based on Vos (1999b).
Probability of CHF post AMI	0.2	Vos, 1999a (WHO Burden of Disease Study, based on Cowie et al., 1997)
Probability of angina post AMI	0.51	American Heart Association, 2003 (Calculated as the population with angina divided by the total population with heart disease)
Quality-of-life preference score for post-AMI with CHF (no angina)	0.80 – 0.89	Uniform distribution with lower bound at 0.80 (Stinnett et al., 1996) and upper bound at 0.89 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with CHF and angina	0.76 – 0.85	Uniform distribution with lower bound at 0.76 (Stinnett et al., 1996, adjusted for severity) and upper bound at 0.85 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with angina (no CHF)	0.7 – 0.89	Uniform distribution with lower bound at 0.7, based on the standard gamble elicitation method (Pliskin, Stason, and Weinstein, 1981) and upper bound at 0.89, based on the time trade-off method (Kuntz et al., 1996).
Quality-of-life preference score for post-AMI (no angina, no CHF)	0.93	Only one value available from the literature. Thus, no distribution is specified. Source of value is Kuntz et al. (1996).

CB is assumed to persist for the remainder of an affected individual's lifespan. Duration of CB will thus equal life expectancy conditioned on having CB. CDC has estimated that COPD (of which CB is one element) results in an average loss of life years equal to 4.26 per COPD death, relative to a reference life expectancy of 75 years (CDC, 2003). Thus, we subtract 4.26 from the remaining life expectancy for each age group, up to age 75. For age groups over 75, we apply the ratio of 4.26 to the life expectancy for the 65 to 74 year group (0.237) to the life expectancy for the 75 to 84 and 85 and up age groups to estimate potential life years lost and then subtract that value from the base life expectancy.

**Table G-5: Estimated Reduction in Incidence of Chronic Bronchitis Associated with Illustrative Attainment Strategies for the Revised and More Stringent Alternative PM NAAQS in 2020**

<i>Age Interval</i>	<i>Reduction in Incidence (95% Confidence Interval)</i>	
	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>
25 – 34	490 (47 – 940)	830 (77 – 1,600)
35 – 44	560 (53 – 1,100)	950 (88 – 1,800)
45 – 54	510 (48 – 960)	880 (81 – 1,700)
55 – 64	490 (46 – 940)	890 (82 – 1,700)
65 – 74	340 (32 – 640)	630 (58 – 1,200)
75 – 84	170 (16 – 320)	310 (28 – 580)
85+	74 (7 – 140)	130 (12 – 250)
Total	2,600 (250 – 5,000)	4,600 (426 – 8,800)

Quality of life with chronic lung diseases has been examined in several studies. In an analysis of the impacts of environmental exposures to contaminants, de Hollander et al. (1999) assigned a weight of 0.69 to years lived with CB. This weight was based on physicians' evaluations of health states similar to CB. Salomon and Murray (2003) estimated a pooled weight of 0.77 based on visual analogue scale, time trade-off, standard gamble, and person trade-off techniques applied to a convenience sample of health professionals. The Harvard Center for Risk Analysis catalog of preference scores reports a weight of 0.40 for severe COPD, with a range from 0.2 to 0.8, based on the judgments of the study's authors (Bell et al., 2001). The Victoria Burden of Disease (BoD) study used a weight of 0.47 for severe COPD and 0.83 for mild to moderate COPD, based on an analysis by Stouthard et al. (1997) of chronic diseases in Dutch populations (Vos, 1999a). Based on the recommendations of Gold et al. (1996), quality-of-life weights based on community preferences are preferred for CEA of interventions affecting broad populations. Use of weights based on health professionals is not recommended. It is not clear from the Victoria BoD study whether the weights used for COPD are based on community preferences or judgments of health professionals. The Harvard catalog score is clearly identified as based on author judgment. Given the lack of a clear preferred weight, we select a triangular distribution centered at 0.7 with an upper bound at 0.9 (slightly better than a mild/moderate case defined by the Victoria BoD study) and a lower bound at 0.5 based on the Victoria BoD study. We will need additional empirical data on quality of life with chronic respiratory diseases based on community preferences to improve our estimates.

Selection of a reference weight for the general population without CB is somewhat uncertain. It is clear that the general population is not in perfect health; however, there is some uncertainty as

to whether individuals' ratings of health states are in reference to a perfect health state or to a generally achievable "normal" health state given age and general health status. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). Following Carrothers, Evans, and Graham (2002), we assumed that the reference weight for the general population without CB is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0. Note that the reference weight for the general population is used solely to determine the incremental quality-of-life improvement applied to the duration of life that would have been lived with the chronic disease. For example, if CB has a quality-of-life weight of 0.7 relative to a reference quality-of-life weight of 0.9, then the incremental quality-of-life improvement is 0.2. If the reference quality-of-life weight is 0.95, then the incremental quality-of-life improvement is 0.25. As noted above, the population is assumed to have a reference weight of 1.0 for all life years gained due to mortality risk reductions.

We present discounted QALYs over the duration of the lifespan with CB using a 3 percent discount rate. Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALYs gained per incidence of CB for each age interval.<sup>4</sup> Based on the assumptions defined above, the mean 3 percent discounted QALY gained per incidence of CB for each age interval along with the 95 percent confidence interval resulting from the Monte Carlo simulation is presented in Table G-6. Table G-6 presents both the undiscounted and discounted QALYs gained per incidence.

**Table G-6: QALYs Gained per Avoided Incidence of CB**

<i>Age Interval</i>		<i>QALYs Gained per Incidence</i>	
<i>Start Age</i>	<i>End Age</i>	<i>Undiscounted</i>	<i>Discounted (3%)</i>
25	34	12.15 (4.40-19.95)	6.52 (2.36-10.71)
35	44	9.91 (3.54-16.10)	5.94 (2.12-9.66)
45	54	7.49 (2.71-12.34)	5.03 (1.82-8.29)
55	64	5.36 (1.95-8.80)	4.03 (1.47-6.61)
65	74	3.40 (1.22-5.64)	2.84 (1.02-4.71)
75	84	2.15 (0.77-3.49)	1.92 (0.69-3.13)
85+		0.79 (0.27-1.29)	0.77 (0.26-1.25)

<sup>4</sup> Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables. For more details, see Gentile (1998).

### G.5.2 Calculating QALYs Associated with Reductions in the Incidence of Nonfatal Myocardial Infarctions

Nonfatal heart attacks, or acute myocardial infarctions, require more complicated calculations to derive estimates of QALY impacts. The actual heart attack, which results when an area of the heart muscle dies or is permanently damaged because of oxygen deprivation, and subsequent emergency care are of relatively short duration. Many heart attacks result in sudden death. However, for survivors, the long-term impacts of advanced CHD are potentially of long duration and can result in significant losses in quality of life and life expectancy.

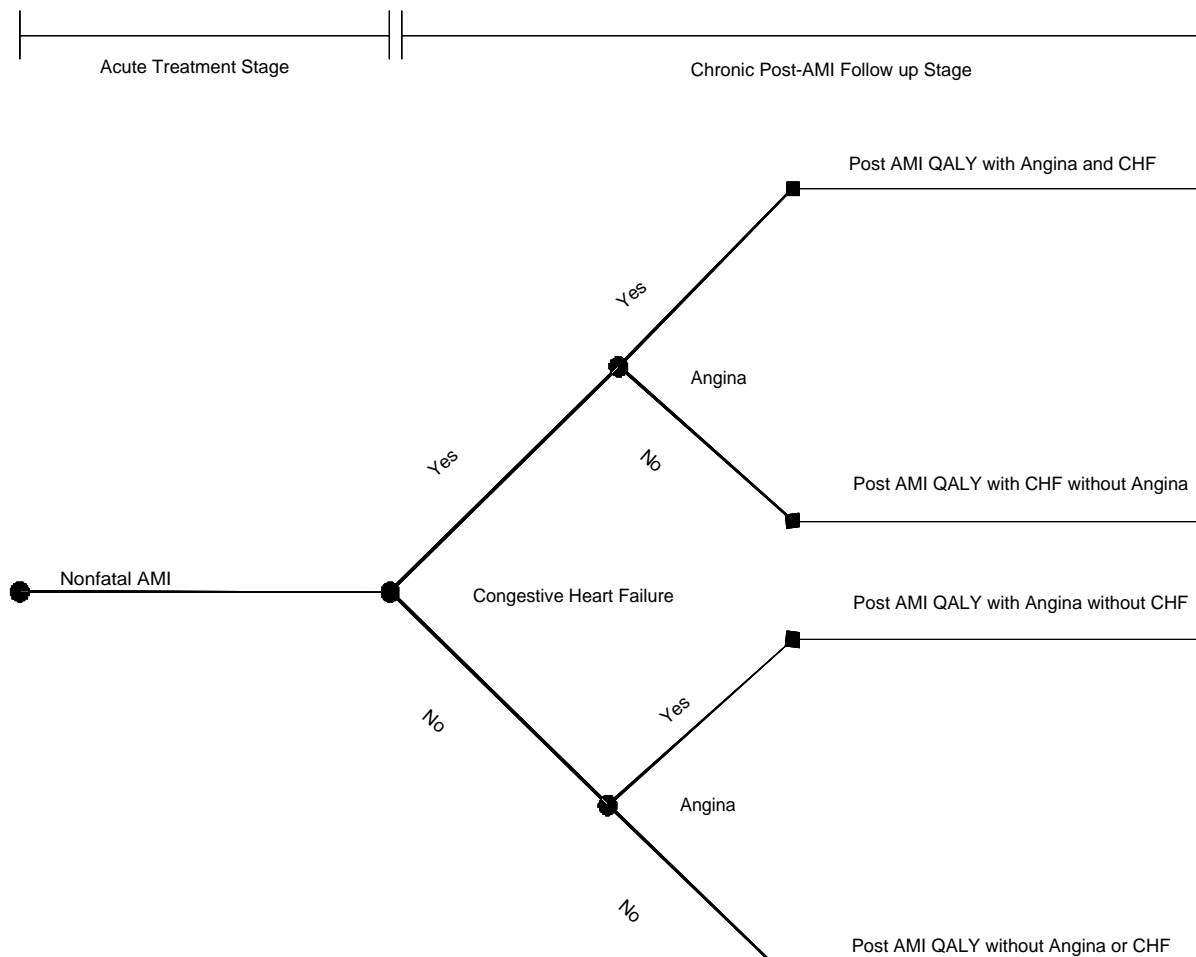
In this phase of the analysis, we did not independently estimate the gains in life expectancy associated with reductions in nonfatal heart attacks. Based on recommendations from the SAB-HES, we assumed that all gains in life expectancy are captured in the estimates of reduced mortality risk provided by the Pope et al. (2002) analysis. We only estimate the change in quality of life over the period of life affected by the occurrence of a heart attack. This may understate the QALY impacts of nonfatal heart attacks but ensures that the overall QALY impact estimates across endpoints do not double-count potential life-year gains.

Our approach adapts a CHD model developed for the Victoria Burden of Disease study (Vos, 1999b). This model accounts for the lost quality of life during the heart attack and the possible health states following the heart attack. Figure G-1 shows the heart attack QALY model in diagrammatic form.

The total gain in QALYs is calculated as:

$$\text{DISCOUNTED AMI QALY GAINED} = \sum_i \Delta \text{AMI}_i \times D_i^{*AMI} \times (w_i - w_i^{AMI}) + \sum_i \sum_{j=1}^4 \Delta \text{AMI}_i \times p_j D_{ij}^{*PostAMI} \times (w_i - w_{ij}^{PostAMI})$$

where  $\Delta \text{AMI}_i$  is the number of nonfatal acute myocardial infarctions avoided in age interval  $i$ ,  $w_i^{AMI}$  is the QALY weight associated with the acute phase of the AMI,  $p_j$  is the probability of being in the  $j$ th post-AMI status,  $w_{ij}^{PostAMI}$  is the QALY weight associated with post-AMI health status  $j$ ,  $w_i$  is the average QALY weight for age interval  $i$ ,  $D_i^{*AMI} = \int_{t=1}^{D_i^{AMI}} e^{-rt} dt$ , the discounted value of  $D_i^{AMI}$ , the duration of the acute phase of the AMI, and  $D_{ij}^{*PostAMI} = \int_{t=1}^{D_{ij}^{PostAMI}} e^{-rt} dt$ , is the discounted value of  $D_{ij}^{PostAMI}$ , the duration of post-AMI health status  $j$ .



**Figure G-1.** Decision Tree Used in Modeling Gains in QALYs from Reduced Incidence of Nonfatal Acute Myocardial Infarctions

Nonfatal heart attacks have been linked with short-term exposures to  $PM_{2.5}$  in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between  $PM_{2.5}$  and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we chose to provide a separate estimate for nonfatal heart attacks based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. These studies provide a weight of evidence for this type of effect. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other CHDs (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997, Tsuji et al., 1996). As such,

significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

The number of avoided nonfatal AMI in each age interval is derived from applying the impact function from Peters et al. (2001) to the population in each age interval with the appropriate baseline incidence rate.<sup>5</sup> The effect estimate from the Peters et al. (2001) study is 0.0241, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.27 for a 10 µg change in PM<sub>2.5</sub>. Table G-7 presents the estimated reduction in nonfatal AMI associated with the illustrative PM NAAQS attainment strategies.

**Table G-7: Estimated Reduction in Nonfatal Acute Myocardial Infarctions Associated with Illustrative Attainment Strategies for the Revised and More Stringent Alternative PM NAAQS in 2020**

<i>Age Interval</i>	<i>Reduction in Incidence*(95% Confidence Interval)</i>	
	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>
18 – 24	1 (1 – 2)	4 (2 – 6)
25 – 34	8 (4 – 12)	26 (13 – 40)
35 – 44	170 (84 – 250)	280 (140 – 430)
45 – 54	520 (260 – 790)	930 (460 – 1,400)
55 – 64	1,300 (630 – 1,900)	2,100 (1,100 – 3,200)
65 – 74	1,500 (770 – 2,300)	2,600 (1,300 – 3,900)
75 – 84	980 (490 – 1,500)	1,800 (900 – 2,800)
85+	520 (260 – 780)	940 (460 – 1,400)
Total	5,000 (2,500 – 7,500)	8,700 (4,300 – 13,000)

Acute myocardial infarction results in significant loss of quality of life for a relatively short duration. The WHO Global Burden of Disease study, as reported in Vos (1999b), assumes that the acute phase of an acute myocardial infarction lasts for 0.06 years, or around 22 days. An alternative assumption is the acute phase is characterized by the average length of hospital stay for an AMI in the United States, which is 5.5 days, based on data from the Agency for

<sup>5</sup> Daily nonfatal myocardial infarction incidence rates per person were obtained from the 1999 National Hospital Discharge Survey (assuming all diagnosed nonfatal AMI visit the hospital). Age-specific rates for four regions are used in the analysis. Regional averages for populations 18 and older are 0.0000159 for the Northeast, 0.0000135 for the Midwest, 0.0000111 for the South, and 0.0000100 for the West.

Healthcare Research and Quality's Healthcare Cost and Utilization Project (HCUP).<sup>6</sup> We assumed a distribution of acute phase duration characterized by a uniform distribution between 5.5 and 22 days, noting that due to earlier discharges and in-home therapy available in the United States, duration of reduced quality of life may continue after discharge from the hospital. In the period during and directly following an AMI (the acute phase), we assigned a quality of life weight equal to 0.605, consistent with the weight for the period in treatment during and immediately after an attack (Vos, 1999b).

During the post-AMI period, a number of different health states can determine the loss in quality of life. We chose to classify post-AMI health status into four states defined by the presence or absence of angina and congestive heart failure (CHF). This makes a very explicit assumption that without the occurrence of an AMI, individuals would not experience either angina or CHF. If in fact individuals already have CHF or angina, then the quality of life gained will be overstated. We do not have information about the percentage of the population have been diagnosed with angina or CHF with no occurrence of an AMI. Nor do we have information on what proportion of the heart attacks occurring due to PM exposure are first heart attacks versus repeat attacks. Probabilities for the four post-AMI health states sum to one.

Given the occurrence of a nonfatal AMI, the probability of congestive heart failure is set at 0.2, following the heart disease model developed by Vos (1999b). The probability is based on a study by Cowie et al. (1997), which estimated that 20 percent of those surviving AMI develop heart failure, based on an analysis of the results of the Framingham Heart Study.

The probability of angina is based on the prevalence rate of angina in the U.S. population. Using data from the American Heart Association, we calculated the prevalence rate for angina by dividing the estimated number of people with angina (6.6 million) by the estimated number of people with CHD of all types (12.9 million). We then assumed that the prevalence of angina in the population surviving an AMI is similar to the prevalence of angina in the total population with CHD. The estimated prevalence rate is 51 percent, so the probability of angina is 0.51.

Combining these factors leads to the probabilities for each of the four health states as follows:

- I. Post AMI with CHF and angina = 0.102
- II. Post AMI with CHF without angina = 0.098
- III. Post AMI with angina without CHF = 0.408
- IV. Post AMI without angina or CHF = 0.392

Duration of post-AMI health states varies, based in part on assumptions regarding life expectancy with post-AMI complicating health conditions. Based on the model used for established market economies (EME) in the WHO Global Burden of Disease study, as reported in Vos (1999b), we assumed that individuals with CHF have a relatively short remaining life expectancy and thus a relatively short period with reduced quality of life (recall that gains in life expectancy are assumed to be captured by the cohort estimates of reduced mortality risk).

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<sup>6</sup> Average length of stay estimated from the HCUP data includes all discharges, including those due to death. As such, the 5.5-day average length of stay is likely an underestimate of the average length of stay for AMI admissions where the patient is discharged alive.

Table G-8 provides the duration (both discounted and undiscounted) of CHF assumed for post-AMI cases by age interval.

**Table G-8: Assumed Duration of Congestive Heart Failure**

<i>Age Interval</i>		<i>Duration of Heart Failure (years)</i>	
<i>Start Age</i>	<i>End Age</i>	<i>Undiscounted</i>	<i>Discounted (3%)</i>
18	24	7.11	6.51
25	34	6.98	6.40
35	44	6.49	6.00
45	54	5.31	4.99
55	64	1.96	1.93
65	74	1.71	1.69
75	84	1.52	1.50
85+		1.52	1.50

Duration of health states without CHF is assumed to be equal to the life expectancy of individuals conditional on surviving an AMI. Ganz et al. (2000) note that “Because patients with a history of myocardial infarction have a higher chance of dying of CHD that is unrelated to recurrent myocardial infarction (for example, arrhythmia), this cohort has a higher risk for death from causes other than myocardial infarction or stroke than does an unselected population.” They go on to specify a mortality risk ratio of 1.52 for mortality from other causes for the cohort of individuals with a previous (nonfatal) AMI. The risk ratio is relative to all-cause mortality for an age-matched unselected population (i.e., general population). We adopted the same ratios and applied them to each age-specific all-cause mortality rate to derive life expectancies (both discounted and undiscounted) for each age group after an AMI, presented in Table G-9. These life expectancies are then used to represent the duration of non-CHF post-AMI health states (III and IV).

**Table G-9: Assumed Duration of Non-CHF Post-AMI Health States**

<i>Age Interval</i>		<i>Post-AMI Years of Life Expectancy (non-CHF)</i>	
<i>Start Age</i>	<i>End Age</i>	<i>Undiscounted</i>	<i>Discounted (3%)</i>
18	24	55.5	27.68
25	34	46.1	25.54
35	44	36.8	22.76
45	54	27.9	19.28
55	64	19.8	15.21
65	74	12.8	10.82
75	84	7.4	6.75
85+		3.6	3.47



For the four post-AMI health states, we used QALY weights based on preferences for the combined conditions characterizing each health state. A number of estimates of QALY weights are available for post-AMI health conditions.

The first two health states are characterized by the presence of CHF, with or without angina. The Harvard Center for Risk Analysis catalog of preference scores provides several specific weights for CHF with and without mild or severe angina and one set specific to post-AMI CHF. Following the Victoria Burden of Disease model, we assumed that most cases of angina will be treated and thus kept at a mild to moderate state. We thus focused our selection on QALY weights for mild to moderate angina. The Harvard database includes two sets of community preference-based scores for CHF (Stinnett et al., 1996; Kuntz et al., 1996). The scores for CHF with angina range from 0.736 to 0.85. The lower of the two scores is based on angina in general with no delineation by severity. Based on the range of the scores for mild to severe cases of angina in the second study, one can infer that an average case of angina has a score around 0.96 of the score for a mild case. Applying this adjustment raises the lower end of the range of preference scores for a mild case of angina to 0.76. We selected a uniform distribution over the range 0.76 to 0.85 for CHF with mild angina, with a midpoint of 0.81. The same two studies in the Harvard catalog also provide weights for CHF without angina. These scores range from 0.801 to 0.89. We selected a uniform distribution over this range, with a midpoint of 0.85.

The third health state is characterized by angina, without the presence of CHF. The Harvard catalog includes five sets of community preference-based scores for angina, one that specifies scores for both mild and severe angina (Kuntz et al., 1996), one that specifies mild angina only (Pliskin, Stason, and Weinstein, 1981), one that specifies severe angina only (Cohen, Breall, and Ho, 1994), and two that specify angina with no severity classification (Salkeld, Phongsavan, and Oldenburg, 1997; Stinnett et al., 1996). With the exception of the Pliskin, Stason, and Weinstein score, all of the angina scores are based on the time trade-off method of elicitation. The Pliskin, Stason, and Weinstein score is based on the standard gamble elicitation method. The scores for the nonspecific severity angina fall within the range of the two scores for mild angina specifically. Thus, we used the range of mild angina scores as the endpoints of a uniform distribution. The range of mild angina scores is from 0.7 to 0.89, with a midpoint of 0.80.

For the fourth health state, characterized by the absence of CHF and/or angina, there is only one relevant community preference score available from the Harvard catalog. This score is 0.93, derived from a time trade-off elicitation (Kuntz et al., 1996). Insufficient information is available to provide a distribution for this weight; therefore, it is treated as a fixed value.

Similar to CB, we assumed that the reference weight for the general population without AMI is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0.

Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALYs gained per incidence of nonfatal AMI for each age interval. For the Monte Carlo simulation, all distributions were assumed to be independent. The mean QALYs gained per incidence of

nonfatal AMI for each age interval is presented in Table G-10, along with the 95 percent confidence interval resulting from the Monte Carlo simulation. Table G-10 presents both the undiscounted and discounted QALYs gained per incidence.

**Table G-10: QALYs Gained per Avoided Nonfatal Myocardial Infarction**

<i>Age Interval</i>		<i>QALYs Gained per Incidence<sup>a</sup></i>	
<i>Start Age</i>	<i>End Age</i>	<i>Undiscounted</i>	<i>Discounted (3%)</i>
18	24	4.18 (1.24-7.09)	2.17 (0.70-3.62)
25	34	3.48 (1.09-5.87)	2.00 (0.68-3.33)
35	44	2.81 (0.88-4.74)	1.79 (0.60-2.99)
45	54	2.14 (0.67-3.61)	1.52 (0.51-2.53)
55	64	1.49 (0.42-2.52)	1.16 (0.34-1.95)
65	74	0.97 (0.30-1.64)	0.83 (0.26-1.39)
75	84	0.59 (0.20-0.97)	0.54 (0.19-0.89)
85+		0.32 (0.13-0.50)	0.31 (0.13-0.49)

<sup>a</sup> Mean of Monte Carlo generated distribution; 95% confidence interval presented in parentheses.

## G.6 Cost-Effectiveness Analysis

Given the estimates of changes in life expectancy and quality of life, the next step is to aggregate life expectancy and quality-of-life gains to form an effectiveness measure that can be compared to costs to develop cost-effectiveness ratios. This section discusses the proper characterization of the combined effectiveness measure and the appropriate calculation of the numerator of the cost-effectiveness ratio.

### G.6.1 Aggregating Life Expectancy and Quality-of-Life Gains

To develop an integrated measure of changes in health, we simply sum together the gains in life years from reduced mortality risk in each age interval with the gains in QALYs from reductions in incidence of CB and acute myocardial infarctions. The resulting measure of effectiveness then forms the denominator in the cost-effectiveness ratio. What is this combined measure of effectiveness? It is not a QALY measure in a strict sense, because we have not adjusted life-expectancy gains for preexisting health status (quality of life). It is however, an effectiveness measure that adds to the standard life years calculation a scaled morbidity equivalent. Thus, we term the aggregate measure morbidity inclusive life years, or MILYs. Alternatively, the combined measure could be considered as QALYs with an assumption that the community preference weight for all life-expectancy gains is 1.0. If one considers that this weight might be

considered to be a “fair” treatment of those with preexisting disabilities, the effectiveness measure might be termed “fair QALY” gained. However, this implies that all aspects of fairness have been addressed, and there are clearly other issues with the fairness of QALYs (or other effectiveness measures) that are not addressed in this simple adjustment. The MILY measure violates some of the properties used in deriving QALY weights, such as linear substitution between quality of life and quantity of life. However, in aggregating life expectancy and quality-of-life gains, it merely represents an alternative social weighting that is consistent with the spirit of the recent OMB guidance on CEA. The guidance notes that “fairness is important in the choice and execution of effectiveness measures” (OMB, 2003). The resulting aggregate measure of effectiveness will not be consistent with a strict utility interpretation of QALYs; however, it may still be a useful index of effectiveness.

Applying the life expectancies and distributions of QALYs per incidence for CB and AMI to estimated distributions of incidences yields distributions of life expectancy and QALYs gained due to the PM NAAQS illustrative attainment strategies. These distributions reflect both the quantified uncertainty in incidence estimates and the quantified uncertainty in QALYs gained per incidence.

For the attainment strategy for the revised 15/35 standards, Table G-11 presents the mean 3 percent discounted MILYs gained for each age interval, broken out by life expectancy and quality-of-life categories. Note that quality-of-life gains occur from age 18 and up, while life expectancy gains accrue only after age 29. This is based on the ages of the study populations in the underlying epidemiological studies. It is unlikely that such discontinuities exist in reality, but to avoid overstating effectiveness, we chose to limit the life-expectancy gains to those occurring in the population 30 and over and the morbidity gains to the specific adult populations examined in the studies. Table G-12 provides the same information for the 14/35 attainment strategy.

It is worth noting that around a third of mortality-related benefits are due to reductions in premature deaths among those 75 and older, while only 7 percent of morbidity benefits occur in this age group. This is due to two factors: (1) the relatively low baseline mortality rates in populations under 75, and (2) the relatively constant baseline rates of chronic disease coupled with the relatively long period of life that is lived with increased quality of life without CB and advanced heart disease.

The relationship between age and the distribution of MILYs gained from mortality and morbidity is shown for the 15/35 attainment strategy in Figure G-2 (the relationship is almost identical for the 14/35 attainment strategy). Because the baseline mortality rate is increasing in age at a much faster rate than the prevalence rate for CB, the share of MILYs gained accounted for by mortality is proportional to age. At the oldest age interval, avoiding incidences of CB leads to only a few MILYs gained, due to the lower number of years lived with CB. MILYs gained from avoided premature mortality is low in the youngest age intervals because of the low overall mortality rates in these intervals, although the number of MILYs per incidence is high. In later years, even though the MILYs gained per incidence avoided is low, the number of cases is very high due to higher baseline mortality rates.

**Table G-11. Estimated Gains in 3 Percent Discounted MILYs Associated with Illustrative Attainment Strategies for the Revised PM NAAQS (15/35) in 2020<sup>a</sup>**

Age	Life Years Gained from Mortality Risk Reductions (95% CI)	QALY Gained from Reductions in Chronic Bronchitis (95% CI)	QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)	Total Gain in MILYs (95% CI)
18–24	—	—	3 (0 – 5)	3 (0 – 5)
25–34	580 (170 – 1,000)	3,200 (240 – 7,600)	15 (4 – 32)	3,800 (810 – 8,200)
35–44	1,700 (600 – 2,900)	3,300 (260 – 7,700)	290 (78 – 600)	5,300 (1,900 – 9,900)
45–54	3,000 (970 – 5,000)	2,600 (210 – 6,000)	770 (210 – 1,600)	6,300 (3,000 – 10,000)
55–64	5,800 (1,900 – 9,800)	2,000 (170 – 4,600)	1,400 (360 – 3,000)	9,200 (4,600 – 14,000)
65–74	6,800 (2,200 – 11,000)	960 (83 – 2,300)	1,200 (320 – 2,600)	9,000 (4,100 – 14,000)
75–84	5,400 (1,800 – 9,100)	320 (28 – 770)	510 (140 – 1,000)	6,200 (2,600 – 10,000)
85+	2,900 (940 – 4,900)	56 (5 – 130)	150 (45 – 300)	3,100 (1,200 – 5,100)
Total	26,000 (18,000 – 34,000)	12,000 (1,100 – 29,000)	4,400 (1,200 – 9,100)	43,000 (28,000 – 62,000)

<sup>a</sup> Note that all estimates have been rounded to two significant digits.

Summing over the age intervals provides estimates of total MILYs gained for the PM NAAQS illustrative attainment strategies. The total number of discounted (3 percent) MILYs gained for the 15/35 attainment strategy is 43,000 (95% CI: 28,000 – 62,000) and for the 14/35 attainment strategy is 75,000 (95% CI: 48,000 – 110,000).

#### *G.6.2 Dealing with Acute Health Effects and Nonhealth Effects*

Health effects from exposure to particulate air pollution encompass a wide array of chronic and acute conditions in addition to premature mortality (EPA, 1996). Although chronic conditions and premature mortality generally account for the majority of monetized benefits, acute symptoms can affect a broad population or sensitive populations (e.g., asthma exacerbations in asthmatic children. In addition, reductions in air pollution may result in a broad set of nonhealth environmental benefits, including improved visibility in national parks, increased agricultural and forestry yields, reduced acid damage to buildings, and a host of other impacts. QALYs address only health impacts, and the OMB guidance notes that “where regulation may yield several different beneficial outcomes, a cost-effectiveness comparison becomes more difficult to interpret because there is more than one measure of effectiveness to incorporate in the analysis.”

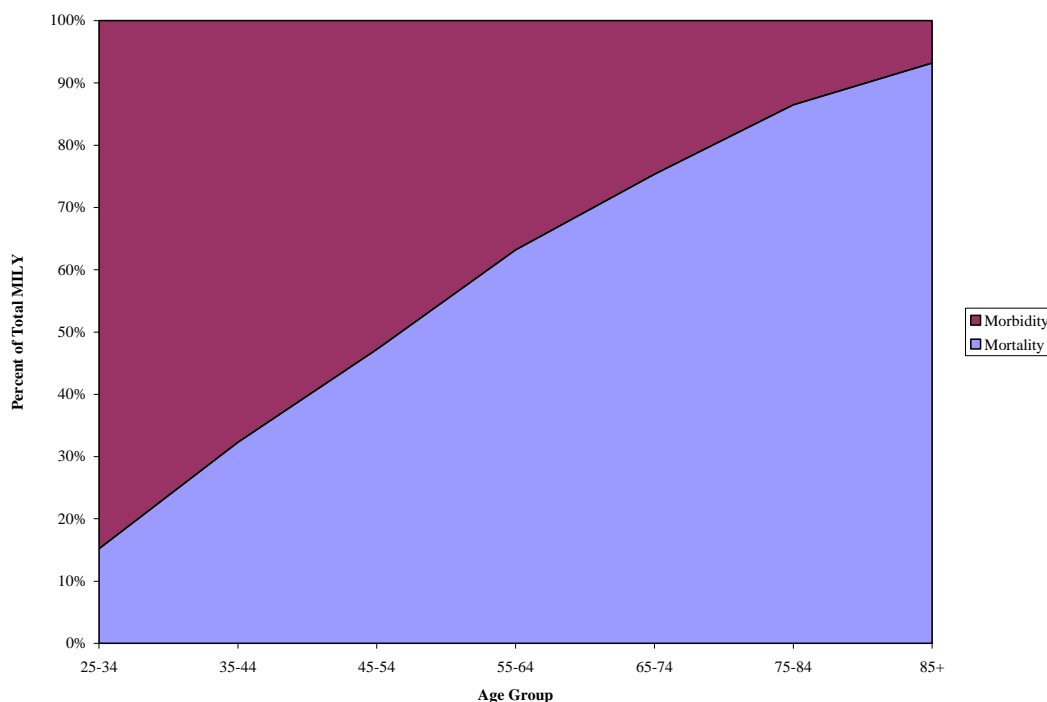
**Table G-12: Estimated Gains in 3 Percent Discounted MILYs Associated with Illustrative Attainment Strategies for the More Stringent Alternative PM NAAQS (14/35) in 2020<sup>a</sup>**

<i>Age</i>	<i>Life Years Gained from Mortality Risk Reductions (95% CI)</i>	<i>QALY Gained from Reductions in Chronic Bronchitis (95% CI)</i>	<i>QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)</i>	<i>Total Gain in MILYs (95% CI)</i>
18–24	—	—	8 (2 – 17)	8 (2 – 17)
25–34	950 (310 – 1,600)	5,500 (390 – 13,000)	51 (13 – 100)	6,500 (1,300 – 14,000)
35–44	2,800 (910 – 4,600)	5,600 (310 – 13,000)	500 (130 – 1,000)	8,900 (3,200 – 17,000)
45–54	4,900 (1,600 – 8,300)	4,400 (320 – 10,000)	1,400 (360 – 2,800)	11,000 (5,000 – 18,000)
55–64	10,000 (3,200 – 17,000)	3,600 (280 – 8,400)	2,400 (600 – 5,000)	16,000 (8,000 – 25,000)
65–74	12,000 (3,800 – 21,000)	1,800 (170 – 4,200)	2,100 (520 – 4,200)	16,000 (7,300 – 25,000)
75–84	9,600 (3,200 – 16,000)	590 (38 – 1,400)	960 (250 – 1,900)	11,000 (4,600 – 18,000)
85+	4,800 (1,600 – 8,100)	98 (7 – 230)	280 (80 – 550)	5,200 (2,000 – 8,400)
Total	45,000 (32,000 – 59,000)	22,000 (1,500 – 51,000)	7,700 (2,000 – 16,000)	75,000 (48,000 – 110,000)

<sup>a</sup> Note that all estimates have been rounded to two significant digits.

With regard to acute health impacts, Bala and Zarkin (2000) suggest that QALYs are not appropriate for valuing acute symptoms, because of problems with both measuring utility for acute health states and applying QALYs in a linear fashion to very short duration health states. Johnson and Lievense (2000) suggest using conjoint analysis to get healthy-utility time equivalences that can be compared across acute effects, but it is not clear how these can be combined with QALYs for chronic effects and loss of life expectancy. There is also a class of effects that EPA has traditionally treated as acute, such as hospital admissions, which may also result in a loss of quality of life for a period of time following the effect. For example, life after asthma hospitalization has been estimated with a utility weight of 0.93 (Bell et al., 2001; Kerridge, Glasziou, and Hillman, 1995).

How should these effects be combined with QALYs for chronic and mortality effects? One method would be to convert the acute effects to QALYs; however, as noted above, there are problems with the linearity assumption (i.e., if a year with asthma symptoms is equivalent to 0.7 year without asthma symptoms, then 1 day without asthma symptoms is equivalent to 0.0019 QALY gained). This is troubling from both a conceptual basis and a presentation basis. An alternative approach is simply to treat acute health effects like nonhealth benefits and subtract the dollar value (based on WTP or COI) from compliance costs in the CEA.



**Figure G-2.** Distribution of Mortality and Morbidity Related MILY Across Age Groups for Illustrative Attainment Strategy for the Revised PM NAAQS (3 percent Discount Rate)

To address the issues of incorporating acute morbidity and nonhealth benefits, OMB suggests that agencies “subtract the monetary estimate of the ancillary benefits from the gross cost estimate to yield an estimated net cost.” As with benefit-cost analysis, any unquantified benefits and/or costs should be noted and an indication of how they might affect the cost-effectiveness ratio should be described. We will follow this recommended “net cost” approach in the illustrative exercise, specifically in netting out the benefits of health improvements other than reduced mortality and chronic morbidity, and the benefits of improvements in visibility at national parks (see Chapter 5 for more details on these benefit categories).

### *G.6.3 Cost-Effectiveness Ratios*

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. The estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient PM<sub>2.5</sub> and the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the elements of the numerator are direct costs of controls minus the avoided COI associated with CB and nonfatal AMI. In addition, to account

for the value of reductions in acute health impacts and nonhealth benefits, we net out the monetized value of these benefits from the numerator to yield a “net cost” estimate. For the MILY aggregate effectiveness measure, the denominator is simply the sum of life years gained from increased life expectancy and the sum of QALYs gained from the reductions in CB and nonfatal AMI.

Avoided costs for CB and nonfatal AMI are based on estimates of lost earnings and medical costs.<sup>7</sup> Using age-specific annual lost earnings and medical costs estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a lifetime present discounted value (in 2000\$) due to CB of \$150,542 for someone between the ages of 27 and 44; \$97,610 for someone between the ages of 45 and 64; and \$11,088 for someone over 65. The corresponding age-specific estimates of lifetime present discounted value (in 2000\$) using a 7 percent discount rate are \$86,026, \$72,261, and \$9,030, respectively. These estimates assumed that 1) lost earnings continue only until age 65, 2) medical expenditures are incurred until death, and 3) life expectancy is unchanged by CB.

Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a present discounted value in lost earnings (in 2000\$) over 5 years due to a myocardial infarction of \$8,774 for someone between the ages of 25 and 44, \$12,932 for someone between the ages of 45 and 54, and \$74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2000\$) using a 7 percent discount rate are \$7,855, \$11,578, and \$66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. Thus, we do not include lost earnings in the cost estimates for these age groups.

Two estimates of the direct medical costs of myocardial infarction are used. The first estimate is from Wittels, Hay, and Gotto (1990), which estimated expected total medical costs of MI over 5 years to be \$51,211 (in 1986\$) for people who were admitted to the hospital and survived hospitalization (there does not appear to be any discounting used). Using the CPI-U for medical care, the Wittels estimate is \$109,474 in year 2000\$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using “knowledgeable cardiologists” as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The second estimate is from Russell et al. (1998), which estimated first-year direct medical costs of treating nonfatal myocardial infarction of \$15,540 (in 1995\$), and \$1,051 annually thereafter. Converting to year 2000\$, that would be \$23,353 for a 5-year period (without discounting).

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<sup>7</sup> Gold et al. (1996) recommend not including lost earnings in the cost-of-illness estimates, suggesting that in some cases, they may be already be counted in the effectiveness measures. However, this requires that individuals fully incorporate the value of lost earnings and reduced labor force participation opportunities into their responses to time-tradeoff or standard-gamble questions. For the purposes of this analysis and for consistency with the way costs-of-illness are calculated for the benefit-cost analysis, we have assumed that individuals do not incorporate lost earnings in responses to these questions. This assumption can be relaxed in future analyses with improved understanding of how lost earnings are treated in preference elicitation.

The two estimates from these studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period. We used a simple average of the two 5-year estimates, or \$65,902, and add it to the 5-year opportunity cost estimate. The resulting estimates are given in Table G-13.

**Table G-13: Estimated Costs Over a 5-Year Period (in 2000\$) of a Nonfatal Myocardial Infarction**

<i>Age Group</i>	<i>Opportunity Cost</i>	<i>Medical Cost<sup>a</sup></i>	<i>Total Cost</i>
0 – 24	\$0	\$65,902	\$65,902
25-44	\$8,774 <sup>b</sup>	\$65,902	\$74,676
45 – 54	\$12,253 <sup>b</sup>	\$65,902	\$78,834
55 – 65	\$70,619 <sup>b</sup>	\$65,902	\$140,649
>65	\$0	\$65,902	\$65,902

<sup>a</sup> An average of the 5-year costs estimated by Wittels, Hay, and Gotto (1990) and Russell et al. (1998).

<sup>b</sup> From Cropper and Krupnick (1990), using a 3 percent discount rate.

The total avoided COI by age group associated with the reductions in CB and nonfatal acute myocardial infarctions is provided in Table G-14. Note that the total avoided COI associated with the revised PM NAAQS is \$520 million and is \$1,200 million for the more stringent alternative. Note that this does not include any direct avoided medical costs associated with premature mortality. Nor does it include any medical costs that occur more than 5 years from the onset of a nonfatal AMI. Therefore, this is likely an underestimate of the true avoided COI associated with strategies for attainment of the PM NAAQS.

**Table G-14: Avoided Costs of Illness Associated with Reductions in Chronic Bronchitis and Nonfatal Acute Myocardial Infarctions Associated with Attainment Strategies for the Revised and More Stringent PM NAAQS in 2020**

<i>Age Range</i>	<i>Avoided Cost of Illness (in millions of 2000\$)</i>			
	<i>Chronic Bronchitis</i>		<i>Nonfatal Acute Myocardial Infarction</i>	
	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>	<i>15/35 Attainment Strategy</i>	<i>14/34 Attainment Strategy</i>
18-24	—	—	\$0.1	\$0.3
25-34	\$73	\$120	\$0.6	\$1.9
35-44	\$83	\$140	\$12	\$20
45-54	\$48	\$84	\$40	\$71
55-64	\$47	\$85	\$170	\$290
65-74	\$3.6	\$6.7	\$98	\$160
75-84	\$1.8	\$3.3	\$62	\$120
85+	\$0.8	\$1.4	\$33	\$60
Total	\$260	\$450	\$420	\$730



## G.7 Discount Rate Sensitivity Analysis

A large number of parameters and assumptions are necessary in conducting a CEA. Where appropriate and supported by data, we have included distributions of parameter values that were used in generating the reported confidence intervals. For the assumed discount rate, we felt it more appropriate to examine the impact of the assumption using a sensitivity analysis rather than through the integrated probabilistic uncertainty analysis.

The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the academic community. OMB and EPA guidance require using both a 7 percent rate and a 3 percent rate. In the most recent benefit-cost analyses of air pollution regulations, a 3 and 7 percent discount rate have been adopted in the primary analysis. A 3 percent discount rate reflects a “social rate of time preference” discounting concept. A 3 percent discount rate is also consistent with the recommendations of the NAS panel on CEA (Gold et al., 1996), which suggests that “a real annual (riskless) rate of 3 percent should be used in the Reference Case analysis.” We have also calculated MILYs and the implicit cost thresholds using a 7 percent rate consistent with an “opportunity cost of capital” concept to reflect the time value of resources directed to meet regulatory requirements. Further discussion of this topic appears in Chapter 7 of Gold et al. (1996), in Chapter 6 of the EPA *Guidelines for Economic Analysis*, and in OMB Circular A-4.

**Table G-15: Summary of Results for the Illustrative Attainment Strategies for the Revised and More Stringent PM NAAQS in 2020<sup>a</sup>**

	<i>Result Using 3% Discount Rate (95% Confidence Interval)</i>	
	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>
Life years gained from mortality risk reductions	26,000 (18,000 – 34,000)	45,000 (32,000 – 59,000)
QALY gained from reductions in chronic bronchitis	12,000 (1,100 – 29,000)	22,000 (1,500 – 51,000)
QALY gained from reductions in acute myocardial infarctions	4,400 (1,200 – 9,100)	7,700 (2,000 – 16,000)
Total gain in MILYs	43,000 (28,000 – 62,000)	75,000 (48,000 – 110,000)
Avoided cost of illness		
Chronic bronchitis	\$260 million (\$170 million – \$410 million)	\$450 million (\$290 million – \$700 million)
Nonfatal AMI	\$420 million (\$230 million – \$680 million)	\$730 million (\$400 million – \$1,200 million)
Implementation strategy costs <sup>b</sup>	\$5.4 billion	\$7.0 billion
Net cost per MILY	\$97,000 (\$66,000 – \$150,000)	\$63,000 (\$37,000 – \$85,000)

<sup>a</sup> Consistent with recommendations of Gold et al. (1996), all summary results are reported at a precision level of two significant digits to reflect limits in the precision of the underlying elements.

<sup>b</sup> Costs are the private firm costs of control, as discussed in Chapter 6, and reflect discounting using firm specific costs of capital.

Table G-16 presents a summary of results using the 7 percent discount rate and the percentage difference between the 7 percent results and the base case 3 percent results. Adoption of a 7 percent discount rate decreases the estimated life years and QALYs gained from implementing the PM NAAQS. Adopting a discount rate of 7 percent results in a 35 percent reduction in the estimated total MILYs gained in each year, while the cost per MILY increases by approximately 60 percent.

**Table G-16: Impacts of Using a 7 Percent Discount Rate on Cost Effectiveness Analysis for the Illustrative Attainment Strategies for the Revised and More Stringent PM NAAQS in 2020**

	<i>Result Using 7 Percent Discount Rate</i>		<i>Percentage Change Relative to Result Using 3 Percent Discount Rate</i>	
	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>	<i>15/35 Attainment Strategy</i>	<i>14/35 Attainment Strategy</i>
Life years gained from mortality risk reductions	16,000	29,000	–38%	–35%
QALY gained from reductions in chronic bronchitis	8,100	14,000	–32%	–36%
QALY gained from reductions in acute myocardial infarctions	3,500	6,000	–20%	–22%
Total gain in MILYs	28,000	49,000	–35%	–35%
Avoided cost of illness				
Chronic bronchitis	\$170 million	\$290 million	–35%	–35%
Nonfatal AMI	\$410 million	\$710 million	–3%	–3%
Net cost per MILY	\$160,000	\$100,000	+65%	+59%

## G.8 Conclusions

We calculated the effectiveness of PM NAAQS attainment strategies based on reductions in premature deaths and incidence of chronic disease. We measured effectiveness using several different metrics, including lives saved, life years saved, and QALYs (for improvements in quality of life due to reductions in incidence of chronic disease). We suggested a new metric for aggregating life years saved and improvements in quality of life, morbidity inclusive life years (MILY) which assumes that society assigns a weight of one to years of life extended regardless of preexisting disabilities or chronic health conditions.

CEA of environmental regulations that have substantial public health impacts may be informative in identifying programs that have achieved cost-effective reductions in health impacts and can suggest areas where additional controls may be justified. However, the overall efficiency of a regulatory action can only be judged through a complete benefit-cost analysis that takes into account all benefits and costs, including both health and nonhealth effects. The

benefit-cost analysis for the PM NAAQS attainment strategies, provided in Chapter 9, shows that the attainment strategies we modeled have potentially large net benefits, indicating that implementation of the revised PM NAAQS will likely result in improvements in overall public welfare.

## G.9 References

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